Exhibit 6

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to replace prophylactic immunisation. This shows ignorance of the principles of immunisation, one of the greatest success stories of medicine. Because we are host to a plethora of microbes which undergo constant mutation, frequently giving rise to pathogenic strains, we have immunity systems. These have evolved, by selection of reproductive advantage, to prevent the extinction of Homo sapiens by pestilence. The system makes us all different in immunological reactivity, exemplified by our rejection of allografts. When a new pathogen arises, its effects on us range from subclinical infection, through illness of varying severity, to death.

Both infection and immunisation lead to occasional autoimmune diseases, due to development of forbidden clones of lymphocytes which accidentally cross-react with a host antigen in mistake for a microbial one. The autoimmune diseases are potentially preventable by immunisations with vaccines which lack the host-cross-reactive antigens. This is being pioneered by Kehoe[1], who is developing a vaccine for rheumatic fever which lacks the host-cross-reactive antigens. Success in this research will save our vulnerable Maori children from rheumatic fever.

If measles immunisation is stopped, some children (particularly those with Maori type H genes)[2] will die of measles and some, like my sister-in-law, Romula Macfarlane, will die of autoimmune encephalomyelitis, which is 700 times more common after measles infection than after measles immunisation.[3,4] The argument applies to the other infectious diseases, including poliomyelitis where immunisation has abolished the previous tragic deaths and paralyses. Far from needing less immunisations, we need more, including new ones against AIDS, rheumatic fever, the other autoimmune diseases and the so-called "trivial" virus infections (Coxsackie, echo, adeno).

If measles immunisation is not lasting indefinitely, it simply needs to be repeated. Apart from immunisation's wonderful benefit in saving our loved ones from illness and death, it is the epitome of "cost effective" medicine.

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Skin reactions and terfenadine

Terfenadine is an H_1 receptor antagonist which is structurally dissimilar from other conventional antihistamines.[1] Along with other newer antihistamines (astemizole, loratadine and cetirizine), terfenadine has gained widespread popularity due to the lack of sedation as a side effect[2]. This group of antihistamines is nonsedating because they are lipophobic and hence do not readily cross the blood-brain barrier.[2] Serious cardiac arrhythmias have been associated with terfenadine and astemizole either in overdose or with concomitant administration of macrolide antibiotics or imidazole antibiotics.[3] Apart from this, the side effect profile of antihistamine medicines is generally of a minor nature.[3] Terfenadine has previously been associated with skin reactions, albeit rarely.[4,5] We present a case of terfenadine associated skin reaction.

A 53 year old woman was admitted with a 6 day history of generalised urticarial pruritic rash, especially the trunk and proximal parts of her limbs, itchy eyes and throat and some mild lip and periorbital swelling. She had a previous history of allergy to penicillins, erythromycin, some plant and grass species, and cats. This patient had suffered from a bee sting approximately 1 month previously and another bee sting 1 week prior to admission. Current medication history consisted of conjugated equine oestrogen tablets (Premarin) for 4–6 weeks, and terfenadine 60 mg twice daily commenced 4–7 days prior to the rash. These were hormone replacement therapy and selftreatment for rhinorrhoea respectively.

Treatment with intravenous hydrocortisone and oral promethazine brought little relief. After 2 days, treatment with terfenadine 60 mg twice daily was reintroduced, resulting in worsening of her rash and more swelling. Terfenadine was discontinued. The regimen was changed to loratadine, ranitidine and ketotifen with resolution of her symptoms over a 12 hour period.

Results of investigations showed raised acute phase protein (C-reactive protein = 66 mg/L), but were otherwise unremarkable including normal eosinophil count and C_4 level.

Whilst an anaphylactic reaction has been reported for intravenous administration of conjugated oestrogens,[6] we are unaware of reports of skin reactions to this medication.

Prior to discharge further inquiry revealed that she had had a previous hospital admission for an allergic reaction consisting of rash and painful joints. This had been attributed to Benadryl cough medicine, a proprietary preparation containing the antihistamine diphenhydramine and an expectorant, ammonium citrate.

The latent period between first intake of terfenadine and the onset of skin reactions is reported to be between three and seven days.[4] Based upon the temporal relationship between terfenadine administration and onset of rash, and the worsening of symptoms on rechallenge, we deduce that the most likely cause for this patient's hypersensitivity reaction was terfenadine.

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The potential adverse effects of soybean phytoestrogens in infant feeding

It is well established that soybean products contain the phytoestrogens daidzein and genistein.[1-3] We have measured the levels of these compounds in several soy-based infant formulas available in New Zealand. The quantities recommended by manufacturers for infant feeding provide an intake (per kg body weight) of approximately three to five times as much daidzein and genistein than amounts which disrupt the menstrual cycle when fed to premenopausal women.[4] Exposure to phytoestrogens during soy-formula feeding is cause for considerable concern given the greater susceptibility of neonates to oestrogens and the likely duration of exposure through infancy.

The soy phytoestrogens act by (1) inhibiting the enzyme 17-ß-hyroxysteroid oxidoreductase, type 1, which converts the relatively impotent oestrone to the much more potent oestradiol; (2) occupying the oestrogen receptor, thus acting as antagonists to the naturally-produced oestradiol, inhibiting its effects (this behaviour is similar to that of another oestrogen agonist-antagonist, tamoxifen).[4] The consequent reduction in oestrogenic action appears to have a useful prophylactic effect against many oestrogen-dependent disorders in adults, including mammary and prostatic tumours.[5] However, the same effect is deleterious in infants. Considerable research has shown that adequate oestradiol is necessary for the imprinting and development of many physical, physiological and behavioural characteristics during the neonatal period and infancy.[6-7] Any decrease in the amount of oestradiol available is potentially harmful. Unfortunately, no specific research has investigated the effects of soy on these characteristics in the human infant, although it has been shown that phytoestrogens are absorbed similarly in infants and adults.[8]

It has been claimed that soy-formulas are unlikely to cause harm to infants because they have been used for years without adverse reports (O'Regan, personal communications, 1 February 1995). However, another oestrogen, diethylstilbestrol (DES), was administered extensively to women over three decades before the spectrum of harmful effects appeared, some manifesting themselves only when DES offspring reached adulthood.[9] Furthermore, although many women have consumed soy products without reports of problems, when a definitive experiment was conducted, consumption of 60 g of soy protein per day for 1 month disrupted the menstrual cycle during, and for up to 3 months after, administration.[4] Therefore the argument that no adverse effects were observed, therefore none occurred, is incogent. It is also plausible that harmful effects have occurred but have not been linked to soy consumption.

Other researchers have similar concerns about exposing young infants to phytoestrogens. The introductory paper presented by the USFDA Department of Health at a recent phytoestrogen conference notes 'phytoestrogens have some of the same capabilities to induce developmental toxicity as do other estrogens' and 'given the DES tragedy, it would be foolish to ignore the possibility that some phytoestrogens constitute a developmental hazard'.[10]

The New Zealand Ministry of Health has advised that parents 'continue to feed their infants soy-based milk formula if they have been advised to do so by their health specialists' (O'Regan, personal communications, 29 March 1995). However, soyformulas are available at supermarkets enabling parents to choose them without medical advice. It would be prudent for general sales of soy-formulas to be stopped. Failing this there is a need for information to be made available to both physicians and